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NEWS 1		Web Page URLs for STN Seminar Schedule - N. America
NEWS 2	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
NEWS 12	Apr 08	"Ask CAS" for self-help around the clock
NEWS 13	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 14	Apr 09	ZDB will be removed from STN
NEWS 15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 19	Jun 03	New e-mail delivery for search results now available
NEWS 20	Jun 10	MEDLINE Reload
NEWS 21	Jun 10	PCTFULL has been reloaded

NEWS EXPRESS	February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS INTER	General Internet Information
NEWS LOGIN	Welcome Banner and News Items
NEWS PHONE	Direct Dial and Telecommunication Network Access to STN
NEWS WWW	CAS World Wide Web Site (general information)

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* * * * * * * * * * * * * * * STN Columbus * * * * * * * * * * * * * * * *

FILE 'HOME' ENTERED AT 13:24:01 ON 12 JUN 2002

=> file medline caplus embase biosis

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FULL ESTIMATED COST	0.21	0.21

FILE 'MEDLINE' ENTERED AT 13:24:18 ON 12 JUN 2002

FILE 'CAPLUS' ENTERED AT 13:24:18 ON 12 JUN 2002
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FILE 'EMBASE' ENTERED AT 13:24:18 ON 12 JUN 2002

FILE 'BIOSIS' ENTERED AT 13:24:18 ON 12 JUN 2002

=> s hillman J?/au or Guegler K?/au or Patterson C?/au
L1 3870 HILLMAN J?/AU OR GUEGLER K?/AU OR PATTERSON C?/AU

=> s l1 and protease?
L2 80 L1 AND PROTEASE?

=> s l2 and growth?
L3 15 L2 AND GROWTH?

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4 7 DUP REM L3 (8 DUPLICATES REMOVED)

=> dis l4 1-7 ibib abs kwic

L4 ANSWER 1 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2001:499589 BIOSIS
DOCUMENT NUMBER: PREV200100499589
TITLE: **Growth**-associated **protease** inhibitor heavy chain precursor.
AUTHOR(S): **Hillman, Jennifer L.; Guegler, Karl J.; Patterson, Chandra**
ASSIGNEE: Incyte Genomics, Inc.
PATENT INFORMATION: US 6228991 May 08, 2001
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (May 8, 2001) Vol. 1246, No. 2, pp. No Pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB The invention provides a human **growth**-associated

protease inhibitor heavy chain precursor (GAPIP) and polynucleotides which identify and encode GAPIP. The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders associated with expression of GAPIP.

TI **Growth**-associated **protease** inhibitor heavy chain precursor.

AU **Hillman, Jennifer L.**; **Guegler, Karl J.**; **Patterson, Chandra**

AB The invention provides a human **growth**-associated **protease** inhibitor heavy chain precursor (GAPIP) and polynucleotides which identify and encode GAPIP. The invention also provides expression vectors, host cells,. . .

IT . . .
Molecular Genetics (Biochemistry and Molecular Biophysics); Methods and Techniques; Pharmacology

IT Diseases
GAPIP expression-associated disorders: disease-miscellaneous

IT Chemicals & Biochemicals
growth-associated **protease** inhibitor heavy chain precursor [GAPIP]; polynucleotides

DUPLICATE 1

L4 ANSWER 2 OF 7 MEDLINE
ACCESSION NUMBER: 2001665722 MEDLINE
DOCUMENT NUMBER: 21567998 PubMed ID: 11711494
TITLE: Cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells.
AUTHOR: Nguyen K T; Frye S R; Eskin S G; **Patterson C**; Runge M S; McIntire L V
CORPORATE SOURCE: Department of Bioengineering, Rice University, Houston, Texas, USA..
CONTRACT NUMBER: HL-03658 (NHLBI)
HL-18672 (NHLBI)
HL-57352 (NHLBI)
NS-23327 (NINDS)
SOURCE: HYPERTENSION, (2001 Nov) 38 (5) 1038-43.
Journal code: 7906255. ISSN: 1524-4563.
(Investigators: McIntire L V, Rice U, Houston, TX)
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals; Space Life Sciences
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20011119
Last Updated on STN: 20020131
Entered Medline: 20011207

AB Cyclic strain regulates many vascular smooth muscle cell (VSMC) functions through changing gene expression. This study investigated the effects of cyclic strain on **protease**-activated receptor-1 (PAR-1) expression in VSMCs and the possible signaling pathways involved, on the basis of the hypothesis that cyclic strain would enhance PAR-1 expression, reflecting increased thrombin activity. Uniaxial cyclic strain (1 Hz, 20%) of cells cultured on elastic membranes induced a 2-fold increase in both PAR-1 mRNA and protein levels. Functional activity of PAR-1, as assessed by cell proliferation in response to thrombin, was also increased by cyclic strain. In addition, treatment of cells with antioxidants or an NADPH oxidase inhibitor blocked strain-induced PAR-1 expression. Preincubation of cells with protein kinase inhibitors (staurosporine or Ro 31-8220) enhanced strain-increased PAR-1 expression, whereas inhibitors of NO synthase, tyrosine kinase, and mitogen-activated protein kinases had no effect. Cyclic strain in the presence of basic fibroblast **growth** factor induced PAR-1 mRNA levels beyond the effect of cyclic strain alone, whereas no additive effect was observed between cyclic strain and platelet-derived **growth** factor-AB. Our findings that cyclic strain upregulates PAR-1 mRNA expression but that shear stress downregulates this gene in VSMCs provide an opportunity to elucidate signaling differences by which VSMCs respond to different mechanical forces.

TI Cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells.

AU Nguyen K T; Frye S R; Eskin S G; **Patterson C**; Runge M S; McIntire L V

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CT . . . Gov't, Non-P.H.S.; Support, U.S. Gov't, P.H.S.
Aorta: ME, metabolism
Cell Division: DE, drug effects
Cells, Cultured
Enzyme Inhibitors: PD, pharmacology
Growth Substances: PD, pharmacology
Muscle, Smooth, Vascular: DE, drug effects
*Muscle, Smooth, Vascular: ME, metabolism
Nitric-Oxide Synthase: PH, physiology
Oxidative. . .

CN 0 (Enzyme Inhibitors); 0 (**Growth** Substances); 0 (RNA, Messenger); 0 (Reactive Oxygen Species); 0 (Receptors, Thrombin); 0 (**protease**-activated receptor 1); EC 1.14.13.39 (Nitric-Oxide Synthase); EC 2.7.1.37 (Protein Kinases); EC 3.4.21.5 (Thrombin)

L4 ANSWER 3 OF 7 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:887825 CAPLUS
DOCUMENT NUMBER: 136:148338
TITLE: Cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells
AUTHOR(S): Nguyen, Kytai T.; Frye, Stacie R.; Eskin, Suzanne G.; **Patterson, Cam**; Runge, Marschall S.; McIntire, Larry V.
CORPORATE SOURCE: Department of Bioengineering, Rice University, Houston, TX, 77251-1892, USA
SOURCE: Hypertension (2001), 38(5), 1038-1043
CODEN: HPRTDN; ISSN: 0194-911X
PUBLISHER: Lippincott Williams & Wilkins
DOCUMENT TYPE: Journal

LANGUAGE: English

AB Cyclic strain regulates many vascular smooth muscle cell (VSMC) functions through changing gene expression. This study investigated the effects of cyclic strain on **protease**-activated receptor-1 (PAR-1) expression in VSMCs and the possible signaling pathways involved, on the basis of the hypothesis that cyclic strain would enhance PAR-1 expression, reflecting increased thrombin activity. Uniaxial cyclic strain (1 Hz, 20%) of cells cultured on elastic membranes induced a 2-fold increase in both PAR-1 mRNA and protein levels. Functional activity of PAR-1, as assessed by cell proliferation in response to thrombin, was also increased by cyclic strain. In addn., treatment of cells with antioxidants or an NADPH oxidase inhibitor blocked strain-induced PAR-1 expression. Preincubation of cells with protein kinase inhibitors (staurosporine or Ro 31-8220) enhanced strain-increased PAR-1 expression, whereas inhibitors of NO synthase, tyrosine kinase, and mitogen-activated protein kinases had no effect. Cyclic strain in the presence of basic fibroblast **growth** factor induced PAR-1 mRNA levels beyond the effect of cyclic strain alone, whereas no additive effect was obsd. between cyclic strain and platelet-derived **growth** factor-AB. These findings that cyclic strain upregulates PAR-1 mRNA expression but that shear stress downregulates this gene in VSMCs provide an opportunity to elucidate signaling differences by which VSMCs respond to different mech. forces.

REFERENCE COUNT: 31 THERE ARE 31 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

TI Cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells

AU Nguyen, Kytai T.; Frye, Stacie R.; Eskin, Suzanne G.; **Patterson, Cam**; Runge, Marschall S.; McIntire, Larry V.

AB Cyclic strain regulates many vascular smooth muscle cell (VSMC) functions through changing gene expression. This study investigated the effects of cyclic strain on **protease**-activated receptor-1 (PAR-1) expression in VSMCs and the possible signaling pathways involved, on the basis of the hypothesis that cyclic strain would enhance PAR-1 expression, reflecting increased thrombin activity. Uniaxial cyclic strain (1 Hz, 20%) of cells cultured on elastic membranes induced a 2-fold increase in both PAR-1 mRNA and protein levels. Functional activity of PAR-1, as assessed by cell proliferation in response to thrombin, was also increased by cyclic strain. In addn., treatment of cells with antioxidants or an NADPH oxidase inhibitor blocked strain-induced PAR-1 expression. Preincubation of cells with protein kinase inhibitors (staurosporine or Ro 31-8220) enhanced strain-increased PAR-1 expression, whereas inhibitors of NO synthase, tyrosine kinase, and mitogen-activated protein kinases had no effect. Cyclic strain in the presence of basic fibroblast **growth** factor induced PAR-1 mRNA levels beyond the effect of cyclic strain alone, whereas no additive effect was obsd. between cyclic strain and platelet-derived **growth** factor-AB. These findings that cyclic strain upregulates PAR-1 mRNA expression but that shear stress downregulates this gene in VSMCs provide an opportunity to elucidate signaling differences by which VSMCs respond to different mech. forces.

ST strain stress **protease** activated receptor 1 vascular smooth muscle

IT Receptors
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(PAR-1 (proteinase-activated receptor 1); cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells)

IT Human
Shear stress
Signal transduction, biological
Strain
(cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells)

IT Oxidative stress, biological
(cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells in relation to oxidative stress)

IT Blood vessel
(smooth muscle; cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells)

IT 77106-92-4, NAD(P)H oxidase 80449-02-1, Protein kinase
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells)

IT 106096-93-9, Basic fibroblast **growth** factor
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells in relation to fibroblast **growth** factor)

IT 9002-04-4, Thrombin
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(cyclic strain increases **protease**-activated receptor-1 expression in vascular smooth muscle cells in relation to thrombin)

L4 ANSWER 4 OF 7 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2001109938 MEDLINE
DOCUMENT NUMBER: 20525510 PubMed ID: 11071917
TITLE: Developmental regulation of FKBP65. An ER-localized extracellular matrix binding-protein.
AUTHOR: **Patterson C E**; Schaub T; Coleman E J; Davis E C
CORPORATE SOURCE: Department of Cell Biology, University of Texas Southwestern Medical Center, Dallas, Texas 75390, USA.
CONTRACT NUMBER: HL-60394 (NHLBI)
SOURCE: MOLECULAR BIOLOGY OF THE CELL, (2000 Nov) 11 (11) 3925-35. Journal code: 9201390. ISSN: 1059-1524.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010202

AB FKBP65 (65-kDa FK506-binding protein) is a member of the highly conserved family of intracellular receptors called immunophilins. All have the property of peptidyl-prolyl cis-trans isomerization, and most have been implicated in folding and trafficking events. In an earlier study, we identified that FKBP65 associates with the extracellular matrix protein tropoelastin during its transport through the cell. In the present study, we have carried out a detailed investigation of the subcellular localization of FKBP65 and its relationship to tropoelastin. Using subcellular fractionation, Triton X-114 phase separation, **protease** protection assays, and immunofluorescence microscopy (IF), we have identified that FKBP65 is contained within the lumen of the endoplasmic

reticulum (ER). Subsequent IF studies colocalized FKBP65 with tropoelastin and showed that the two proteins dissociate before reaching the Golgi apparatus. Immunohistochemical localization of FKBP65 in developing lung showed strong staining of vascular and airway smooth muscle cells. Similar areas stained positive for the presence of elastic fibers in the extracellular matrix. The expression of FKBP65 was investigated during development as tropoelastin is not expressed in adult tissues. Tissue-specific expression of FKBP65 was observed in 12-d old mouse tissues; however, the pattern of expression of FKBP65 was not restricted to those tissues expressing tropoelastin. This suggests that additional ligands for FKBP65 likely exist within the ER. Remarkably, in the adult tissues examined, FKBP65 expression was absent or barely detectable. Taken together, these results support an ER-localized FKBP65-tropoelastin interaction that occurs specifically during **growth** and development of tissues.

AU **Patterson C E**; Schaub T; Coleman E J; Davis E C

AB . . . detailed investigation of the subcellular localization of FKBP65 and its relationship to tropoelastin. Using subcellular fractionation, Triton X-114 phase separation, **protease** protection assays, and immunofluorescence microscopy (IF), we have identified that FKBP65 is contained within the lumen of the endoplasmic reticulum. . . . FKBP65 expression was absent or barely detectable. Taken together, these results support an ER-localized FKBP65-tropoelastin interaction that occurs specifically during **growth** and development of tissues.

CT . . .
Reticulum: ME, metabolism
*Extracellular Matrix: ME, metabolism
Gene Expression Regulation, Developmental
Golgi Apparatus: ME, metabolism
Intracellular Membranes
Lung: CY, cytology
***Lung: GD, growth & development**
Lung: ME, metabolism
Mice
Molecular Sequence Data
Muscle, Smooth: ME, metabolism
Tacrolimus Binding Proteins: GE, genetics

. . .

L4 ANSWER 5 OF 7 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:723056 CAPLUS
DOCUMENT NUMBER: 131:333062
TITLE: sequence and therapeutic applications for human **growth**-associated **protease** inhibitor heavy chain precursor
INVENTOR(S): **Hillman, Jennifer L.; Guegler, Karl J.; Patterson, Chandra**
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9957140	A2	19991111	WO 1999-US9947	19990505
WO 9957140	A3	19991229		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6001596	A	19991214	US 1998-74579	19980507
CA 2328132	AA	19991111	CA 1999-2328132	19990505
AU 9938867	A1	19991123	AU 1999-38867	19990505
EP 1078062	A2	20010228	EP 1999-921739	19990505
R: BE, DE, ES, FR, GB, IT, NL				
JP 2002513553	T2	20020514	JP 2000-547109	19990505
US 6228991	B1	20010508	US 1999-388774	19990902

PRIORITY APPLN. INFO.: US 1998-74579 A2 19980507
WO 1999-US9947 W 19990505

AB The invention provides a human **growth**-assocd. **protease** inhibitor heavy chain precursor (GAPIP) and polynucleotides which identify and encode GAPIP. The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders involving the reproductive tract or immunol. disorder or developmental or neoplastic disorders assocd. with expression of GAPIP. An assay for identification of mols. which interact with GAPIP is described.

TI sequence and therapeutic applications for human **growth** -associated **protease** inhibitor heavy chain precursor

IN **Hillman, Jennifer L.; Guegler, Karl J.; Patterson, Chandra**

AB The invention provides a human **growth**-assocd. **protease** inhibitor heavy chain precursor (GAPIP) and polynucleotides which identify and encode GAPIP. The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders involving the reproductive tract or immunol. disorder or developmental or neoplastic disorders assocd. with expression of GAPIP. An assay for identification of mols. which interact with GAPIP is described.

ST therapy sequence human **growth protease** inhibitor heavy chain

IT Nucleic acid hybridization
(DNA-DNA; sequence and therapeutic applications for human **growth**-assocd. **protease** inhibitor heavy chain precursor)

IT Immunity
Reproduction, animal
(disorder; sequence and therapeutic applications for human **growth**-assocd. **protease** inhibitor heavy chain precursor)

IT Antitumor agents
Diagnosis
Drug delivery systems
Genetic vectors
Protein sequences
cDNA sequences

(sequence and therapeutic applications for human **growth**
-assocd. **protease** inhibitor heavy chain precursor)

IT Antibodies
Probes (nucleic acid)
RL: ARG (Analytical reagent use); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(sequence and therapeutic applications for human **growth**
-assocd. **protease** inhibitor heavy chain precursor)

IT 249916-69-6
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid sequence; sequence and therapeutic applications for human **growth**-assocd. **protease** inhibitor heavy chain precursor)

IT 249916-73-2
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide sequence; sequence and therapeutic applications for human **growth**-assocd. **protease** inhibitor heavy chain precursor)

IT 249760-99-4, PN: WO9957140 SEQID: 3 unclaimed protein 249761-00-0, PN: WO9957140 SEQID: 4 unclaimed protein 249761-01-1, PN: WO9957140 SEQID: 5 unclaimed protein
RL: PRP (Properties)
(unclaimed protein sequence; sequence and therapeutic applications for human **growth**-assocd. **protease** inhibitor heavy chain precursor)

L4 ANSWER 6 OF 7 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2000:282931 BIOSIS
DOCUMENT NUMBER: PREV200000282931
TITLE: **Growth**-associated **protease** inhibitor heavy chain precursor.
AUTHOR(S): **Hillman, Jennifer L.; Guegler, Karl J.; Patterson, Chandr**
ASSIGNEE: Incyte Pharmaceuticals, Inc.
PATENT INFORMATION: US 6001596 December 14, 1999
SOURCE: Official Gazette of the United States Patent and Trademark Office Patents, (Dec. 14, 1999) Vol. 1229, No. 2, pp. No pagination. e-file.
ISSN: 0098-1133.
DOCUMENT TYPE: Patent
LANGUAGE: English
AB The invention provides a human **growth**-associated **protease** inhibitor heavy chain precursor (GAPIP) and polynucleotides which identify and encode GAPIP. The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders associated with expression of GAPIP.
TI **Growth**-associated **protease** inhibitor heavy chain precursor.
AU **Hillman, Jennifer L.; Guegler, Karl J.; Patterson, Chandr**
AB The invention provides a human **growth**-associated **protease** inhibitor heavy chain precursor (GAPIP) and polynucleotides which identify and encode GAPIP. The invention also provides expression vectors, host cells,. . .
IT Major Concepts
Enzymology (Biochemistry and Molecular Biophysics); Human Medicine (Medical Sciences)
IT Chemicals & Biochemicals
growth-associated **protease** inhibitor heavy chain precursor [GAPIP]: enzyme inhibitor

L4 ANSWER 7 OF 7 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 89350177 MEDLINE
DOCUMENT NUMBER: 89350177 PubMed ID: 2669631
TITLE: Regulation of neurotoxin and **protease** formation in Clostridium botulinum Okra B and Hall A by arginine.
AUTHOR: **Patterson-Curtis S I**; Johnson E A
CORPORATE SOURCE: Department of Food Microbiology and Toxicology, University of Wisconsin, Madison 53706.
SOURCE: APPLIED AND ENVIRONMENTAL MICROBIOLOGY, (1989 Jun) 55 (6) 1544-8.
Journal code: 7605801. ISSN: 0099-2240.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198909
ENTRY DATE: Entered STN: 19900309
Last Updated on STN: 19970203
Entered Medline: 19890921
AB Supplementation of a minimal medium with high levels of arginine (20 g/liter) markedly decreased neurotoxin titers and **protease** activities in cultures of Clostridium botulinum Okra B and Hall A. Nitrogenous nutrients that are known to be derived from arginine, including proline, glutamate, and ammonia, also decreased **protease** and toxin but less so than did arginine. **Proteases** synthesized during **growth** were rapidly inactivated after **growth** stopped in media containing high levels of arginine. Separation of extracellular proteins by electrophoresis and immunoblots with antibodies to toxin showed that the decrease in toxin titers in media containing high levels of arginine was caused by both reduced synthesis of protoxin and impaired proteolytic activation. In contrast, certain other nutritional conditions stimulated **protease** and toxin formation in C. botulinum and counteracted the repression by arginine. Supplementation of the minimal medium with casein or casein hydrolysates increased **protease** activities and toxin titers. Casein supplementation of a medium containing high levels of arginine prevented **protease** inactivation. High levels of glucose (50 g/liter) also delayed the inactivation of **proteases** in both the minimal medium and a medium containing high levels of arginine. These observations suggest that the availability of nitrogen and energy sources, particularly arginine, affects the production and proteolytic processing of toxins and **proteases** in C. botulinum.
TI Regulation of neurotoxin and **protease** formation in Clostridium botulinum Okra B and Hall A by arginine.
AU **Patterson-Curtis S I**; Johnson E A
AB Supplementation of a minimal medium with high levels of arginine (20 g/liter) markedly decreased neurotoxin titers and **protease** activities in cultures of Clostridium botulinum Okra B and Hall A. Nitrogenous nutrients that are known to be derived from arginine,

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=> dis his

(FILE 'HOME' ENTERED AT 13:24:01 ON 12 JUN 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 13:24:18 ON 12 JUN 2002
L1 3870 S HILLMAN J?/AU OR GUEGLER K?/AU OR PATTERSON C?/AU
L2 80 S L1 AND PROTEASE?
L3 15 S L2 AND GROWTH?
L4 7 DUP REM L3 (8 DUPLICATES REMOVED)

=> end
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LOGOFF? (Y)/N/HOLD:y

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DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-1.24	-1.24

STN INTERNATIONAL LOGOFF AT 13:26:48 ON 12 JUN 2002

Connecting via Winsock to STN

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LOGINID:ssspta1644axd

PASSWORD:
TERMINAL (ENTER 1, 2, 3, OR ?):2

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NEWS 2	Jan 25	BLAST(R) searching in REGISTRY available in STN on the Web
NEWS 3	Jan 29	FSTA has been reloaded and moves to weekly updates
NEWS 4	Feb 01	DKILIT now produced by FIZ Karlsruhe and has a new update frequency
NEWS 5	Feb 19	Access via Tymnet and SprintNet Eliminated Effective 3/31/02
NEWS 6	Mar 08	Gene Names now available in BIOSIS
NEWS 7	Mar 22	TOXLIT no longer available
NEWS 8	Mar 22	TRCTHERMO no longer available
NEWS 9	Mar 28	US Provisional Priorities searched with P in CA/CAplus and USPATFULL
NEWS 10	Mar 28	LIPINSKI/CALC added for property searching in REGISTRY
NEWS 11	Apr 02	PAPERCHEM no longer available on STN. Use PAPERCHEM2 instead.
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NEWS 13	Apr 09	BEILSTEIN: Reload and Implementation of a New Subject Area
NEWS 14	Apr 09	ZDB will be removed from STN
NEWS 15	Apr 19	US Patent Applications available in IFICDB, IFIPAT, and IFIUDB
NEWS 16	Apr 22	Records from IP.com available in CAPLUS, HCAPLUS, and ZCAPLUS
NEWS 17	Apr 22	BIOSIS Gene Names now available in TOXCENTER
NEWS 18	Apr 22	Federal Research in Progress (FEDRIP) now available
NEWS 19	Jun 03	New e-mail delivery for search results now available
NEWS 20	Jun 10	MEDLINE Reload
NEWS 21	Jun 10	PCTFULL has been reloaded
NEWS EXPRESS		February 1 CURRENT WINDOWS VERSION IS V6.0d, CURRENT MACINTOSH VERSION IS V6.0a(ENG) AND V6.0Ja(JP), AND CURRENT DISCOVER FILE IS DATED 05 FEBRUARY 2002
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NEWS PHONE		Direct Dial and Telecommunication Network Access to STN
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FILE 'HOME' ENTERED AT 13:52:16 ON 12 JUN 2002

=> file medline caplus embase biosis

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FILE 'EMBASE' ENTERED AT 13:52:33 ON 12 JUN 2002

FILE 'BIOSIS' ENTERED AT 13:52:33 ON 12 JUN 2002

=> s (protease (1N) inhibitor?)
L1 70323 (PROTEASE (1N) INHIBITOR?)

=> s l1 (10N) growth
L2 912 L1 (10N) GROWTH

=> s l2 (P) human?
L3 212 L2 (P) HUMAN?

=> s l3 (P) sequence?
L4 35 L3 (P) SEQUENCE?

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 15 DUP REM L4 (20 DUPLICATES REMOVED)

=> dis l5 1-15 ibib abs kwic

L5 ANSWER 1 OF 15 MEDLINE DUPLICATE 1
ACCESSION NUMBER: 2002016001 MEDLINE
DOCUMENT NUMBER: 21325286 PubMed ID: 11432724
TITLE: Intracellular location and nuclear targeting of the Spi-1, Spi-2 and Spi-3 gene-derived serine protease inhibitors in non-secretory cells.
AUTHOR: Rothbarth K; Kempf T; Juodka B; Glaser T; Stammer H; Werner D
CORPORATE SOURCE: Division of Biochemistry of the Cell (B0300), German Cancer Research Center, Heidelberg.
SOURCE: EUROPEAN JOURNAL OF CELL BIOLOGY, (2001 May) 80 (5) 341-8. Journal code: 7906240. ISSN: 0171-9335.
PUB. COUNTRY: Germany: Germany, Federal Republic of
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200112
ENTRY DATE: Entered STN: 20020121
Last Updated on STN: 20020121
Entered Medline: 20011204
AB Proteases and their inhibitors are indispensable for the regulated activation and/or degradation of structural and functional proteins involved in basic cellular processes, e.g. in cell cycle control, cell **growth**, differentiation and apoptosis. In this context the serine **protease inhibitors** derived from the murine Spi-1, Spi-2 and Spi-3 genes, and their **human** homologs, deserve reconsideration. Microsequencing data indicate that a fraction of the three serpins has the capability to constitute a well characterized proteinase K, high salt and SDS-stable complex which coisolates with DNA under salting out conditions from various cell and tissue types. This tight association with DNA isolated under conditions designed to deproteinize DNA efficiently points to an in situ preformed chromatin complex. Accordingly, in addition to their well known functions as 'serum protease inhibitors' the Spi-1 and Spi-2 gene-derived proteins appear to have intracellular functions as well. The involvement of the three serpins in chromatin complexes requires their nuclear translocation. Application of (enhanced) green fluorescent protein technology and optical section microscopy reveals that truncation of the N-terminal signal **sequences** of the Spi-1 and Spi-2 gene-encoded proteins is a prerequisite for their nuclear translocation while non-truncated fusion proteins are enriched at the nuclear indentation which is the site of the Golgi apparatus and the centrosome. The identification of new species of intracellular serpins is of potential interest with respect to accumulating evidence for serine protease inhibitor-dependent inhibition or prevention of apoptosis.
AB . . . regulated activation and/or degradation of structural and functional proteins involved in basic cellular processes, e.g. in cell cycle control, cell **growth**, differentiation and apoptosis. In this context the serine **protease inhibitors** derived from the murine Spi-1, Spi-2 and Spi-3 genes, and their **human** homologs, deserve reconsideration. Microsequencing data indicate that a fraction of the three serpins has the capability to constitute a well. . . nuclear translocation. Application of (enhanced) green fluorescent protein technology and optical section microscopy reveals that truncation of the N-terminal signal **sequences** of the Spi-1 and Spi-2 gene-encoded proteins is a prerequisite for their nuclear translocation while non-truncated fusion proteins are enriched. . .

L5 ANSWER 2 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 2002:186414 BIOSIS
DOCUMENT NUMBER: PREV200200186414
TITLE: Analysis of gene expression in a B cell neoplasm with the phenotype of a primary effusion lymphoma (PEL.
AUTHOR(S): Parker, Charles J. (1); Vreeke, Teresa M.; Lin, Chen-Yong; Dickson, Robert B.
CORPORATE SOURCE: (1) Medicine, VA Medical Center, Salt Lake City, UT USA
SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 302a. http://www.bloodjournal.org/. print.
Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11, 2001
ISSN: 0006-4971.
DOCUMENT TYPE: Conference
LANGUAGE: English
AB PEL is defined by the presence of a lymphomatous effusion in the absence of an identifiable contiguous tumor mass. In most instances, the malignant cells are infected with both Epstein Barr virus (EBV) and **human** herpes virus-8 (HHV-8). In a minority of cases, the lymphoma cells are infected with either EBV or HHV-8. When infected with EBV, t(8:14) is invariably present, but this translocation is not observed if the cells are infected with HHV-8. To gain insight into the molecular basis of the unique phenotype, the pattern of gene expression in cells derived from a

patient with the clinical characteristics of PEL was analyzed. The malignant cells were cultured from the pleural fluid of an elderly HIV-negative man. Immunophenotyping was consistent with a lambda light-chain restricted B cell neoplasm of follicular center cell origin. Multiple cytogenetic abnormalities, including t(8:14) were observed in the lymphoma cells. Representation difference analysis (RDA) of DNA revealed fragments of the EBV genome in the tumor cells but HHV-8 DNA was absent by both RDA and PCR analysis. By using RDA modified for cDNA, the pattern of gene expression of the lymphoma cells was compared to that of the patient"s peripheral blood B cells immortalized with EBV. A combination of Southern, northern, and **sequence** analysis showed that 12/40 clones from the tumor sample matched the cDNA of matriptase, a protease that degrades extracellular matrix and is implicated in tumor invasion and metastasis. In addition, matriptase was shown to activate urokinase-type plasminogen activator, hepatocyte growth factor, and protease activated receptor-2. In all tissues examined previously (both normal and malignant), matriptase was invariably co-expressed along with its congnate inhibitor, hepatic **growth** factor activator inhibitor-1 (HAI-1). This Kunitz-type serine **protease inhibitor** is a type I integral membrane protein that regulates matriptase by binding to the activated protein on the cell surface with subsequent shedding of the ectodomain of the complex. Analysis by both flow cytometry and western blot using monoclononal antibodies demonstrated that the PEL cells expressed matriptase protein but not its cognate inhibitor, HAI-1. In contrast, the control cell line expressed HAI-1 but not matriptase. This case appears to be the first reported instance in which matriptase is expressed in the absence of its congnate inhibitor. Because of its potential role in degradation of extracellular matrix, tumor invasion, metastasis, and activation of growth factors and other proteins relevant to the malignant process, we postulate that unregulated expression of matriptase contributed to the unique clinical phenotype of this unusual B cell neoplasm.

AB. . . an identifiable contiguous tumor mass. In most instances, the malignant cells are infected with both Epstein Barr virus (EBV) and **human** herpes virus-8 (HHV-8). In a minority of cases, the lymphoma cells are infected with either EBV or HHV-8. When infected. . . was compared to that of the patient"s peripheral blood B cells immortalized with EBV. A combination of Southern, northern, and **sequence** analysis showed that 12/40 clones from the tumor sample matched the cDNA of matriptase, a protease that degrades extracellular matrix. . . receptor-2. In all tissues examined previously (both normal and malignant), matriptase was invariably co-expressed along with its congnate inhibitor, hepatic **growth** factor activator inhibitor-1 (HAI-1). This Kunitz-type serine **protease inhibitor** is a type I integral membrane protein that regulates matriptase by binding to the activated protein on the cell surface. . .

L5 ANSWER 3 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:727687 CAPLUS
DOCUMENT NUMBER: 135:29659
TITLE: The WFDC1 gene encoding ps20 localizes to 16q24, a region of LOH in multiple cancers
AUTHOR(S): Larsen, Melinda; Ressler, Steven J.; Gerdes, Michael J.; Lu, Bing; Byron, Meg; Lawrence, Jeanne B.; Rowley, David R.
CORPORATE SOURCE: Cell and Molecular Biology Program, Baylor College of Medicine, Houston, TX, 77030, USA
SOURCE: Mammalian Genome (2000), 11(9), 767-773
CODEN: MAMGEC; ISSN: 0938-8990
PUBLISHER: Springer-Verlag New York Inc.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB We previously identified ps20 protein as a secreted growth inhibitor and purified the protein from fetal rat prostate urogenital sinus mesenchymal cell conditioned medium. The rat cDNA was subsequently cloned, and ps20 was found to contain a WAP-type four-disulfide core motif, indicating it may function as a protease inhibitor. We now report cloning and characterization of the mouse ps20 gene (designated Wfdc1), the human homolog cDNA, and the human gene (designated WFDC1). Both the mouse and human WFDC1 genes consist of seven exons and encode resp. ps20 proteins sharing 79.1% identify and nearly identical WAP motifs in exon 2. The WFDC1 gene was mapped by FISH anal. to human Chromosome (Chr) 16q24, an area of frequent loss of heterozygosity (LOH) previously identified in multiple cancers including prostate, breast, hepatocellular, and Wilms' tumor. Identification and characterization of the WFDC1 gene may aid in better understanding the potential role of this gene and ps20 in prostate biol. and carcinogenesis.

REFERENCE COUNT: 45 THERE ARE 45 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

IT Protein motifs
(WAP signature motif; amino acid **sequences** and protein motifs of the **human** and mouse ps20 proteins, a secreted **growth** inhibitor with motifs indicating it may function as a **protease inhibitor**)

IT Protein **sequences**
(amino acid **sequences** and protein motifs of the **human** and mouse ps20 proteins, a secreted **growth** inhibitor with motifs indicating it may function as a **protease inhibitor**)

IT Proteins, specific or class
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(genes Wfdc1 and WFDC1, ps20 (prostate stromal 20); amino acid **sequences** and protein motifs of the **human** and mouse ps20 proteins, a secreted **growth** inhibitor with motifs indicating it may function as a **protease inhibitor**)

IT Growth inhibitors, animal
RL: BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study)
(genes Wfdc1 and WFDC1; amino acid **sequences** and protein motifs of the **human** and mouse ps20 proteins, a secreted **growth** inhibitor with motifs indicating it may function as a **protease inhibitor**)

L5 ANSWER 4 OF 15 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 2000394500 MEDLINE
DOCUMENT NUMBER: 20359916 PubMed ID: 10775503
TITLE: Serpins identified as cell growth inhibitors in human plasma.
AUTHOR: Yao J; Baecher-Allan C M; Sharon J
CORPORATE SOURCE: Department of Pathology and Laboratory Medicine, Boston University School of Medicine, 715 Albany Street, Boston, Massachusetts, 02118, USA.

SOURCE: MOLECULAR CELL BIOLOGY RESEARCH COMMUNICATIONS, (2000 Feb) 3 (2) 76-81.
Journal code: 100889076. ISSN: 1522-4724.

PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200008
ENTRY DATE: Entered STN: 20000824
Last Updated on STN: 20000824
Entered Medline: 20000815

AB We have identified a new factor, CFX, in **human** serum and plasma that inhibits the growth of cultured **human** and mouse cell lines. CFX was determined to be a negatively charged, hydrophobic glycoprotein, with a native molecular weight of 110-120 kDa and a minimal active subunit of 55 kDa. It is precipitated by 60% ammonium sulfate and is resistant to heat treatment at 100 degrees C for 30 min. CFX was purified from **human** plasma to a single band on a gel which retained the cell growth inhibitory activity. Amino acid **sequence** analysis of the CFX band revealed **sequences** from four **human** glycoproteins, alpha1-antichymotrypsin, C1-esterase inhibitor, alpha1-antitrypsin, and alpha2-antiplasmin, all members of the superfamily of serpins. Of the four, C1-esterase inhibitor was shown to be the most potent cell growth inhibitor. These results suggest that serpins may play a cell **growth** inhibitory role in vivo, in addition to their role as **protease inhibitors**.

AB We have identified a new factor, CFX, in **human** serum and plasma that inhibits the growth of cultured **human** and mouse cell lines. CFX was determined to be a negatively charged, hydrophobic glycoprotein, with a native molecular weight of. . . 60% ammonium sulfate and is resistant to heat treatment at 100 degrees C for 30 min. CFX was purified from **human** plasma to a single band on a gel which retained the cell growth inhibitory activity. Amino acid **sequence** analysis of the CFX band revealed **sequences** from four **human** glycoproteins, alpha1-antichymotrypsin, C1-esterase inhibitor, alpha1-antitrypsin, and alpha2-antiplasmin, all members of the superfamily of serpins. Of the four, C1-esterase inhibitor was shown to be the most potent cell growth inhibitor. These results suggest that serpins may play a cell **growth** inhibitory role in vivo, in addition to their role as **protease inhibitors**.
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L5 ANSWER 5 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:723056 CAPLUS
DOCUMENT NUMBER: 131:333062
TITLE: **sequence** and therapeutic applications for **human growth**-associated **protease inhibitor** heavy chain precursor
INVENTOR(S): Hillman, Jennifer L.; Guegler, Karl J.; Patterson, Chandra
PATENT ASSIGNEE(S): Incyte Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 92 pp.
CODEN: PIXXD2

DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9957140	A2	19991111	WO 1999-US9947	19990505
WO 9957140	A3	19991229		
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6001596	A	19991214	US 1998-74579	19980507
CA 2328132	AA	19991111	CA 1999-2328132	19990505
AU 9938867	A1	19991123	AU 1999-38867	19990505
EP 1078062	A2	20010228	EP 1999-921739	19990505
R: BE, DE, ES, FR, GB, IT, NL				
JP 2002513553	T2	20020514	JP 2000-547109	19990505
US 6228991	B1	20010508	US 1999-388774	19990902
PRIORITY APPLN. INFO.:			US 1998-74579 A2	19980507
			WO 1999-US9947 W	19990505

AB The invention provides a human growth-assocd. protease inhibitor heavy chain precursor (GAPIP) and polynucleotides which identify and encode GAPIP. The invention also provides expression vectors, host cells, antibodies, agonists, and antagonists. The invention also provides methods for diagnosing, treating or preventing disorders involving the reproductive tract or immunol. disorder or developmental or neoplastic disorders assocd. with expression of GAPIP. An assay for identification of mols. which interact with GAPIP is described.

TI **sequence** and therapeutic applications for **human growth**-associated **protease inhibitor** heavy chain precursor

ST therapy **sequence human growth protease inhibitor** heavy chain

IT Nucleic acid hybridization
(DNA-DNA; **sequence** and therapeutic applications for **human growth**-assocd. **protease inhibitor** heavy chain precursor)

IT Immunity
Reproduction, animal
(disorder; **sequence** and therapeutic applications for **human growth**-assocd. **protease inhibitor** heavy chain precursor)

IT Antitumor agents
Diagnosis
Drug delivery systems
Genetic vectors
Protein **sequences**
cDNA **sequences**
(**sequence** and therapeutic applications for **human growth**-assocd. **protease inhibitor** heavy chain precursor)

IT Antibodies

Probes (nucleic acid)
RL: ARG (Analytical reagent use); BSU (Biological study, unclassified); ANST (Analytical study); BIOL (Biological study); USES (Uses)
(**sequence** and therapeutic applications for **human growth**-assocd. **protease inhibitor** heavy chain precursor)

IT 249916-69-6
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(amino acid **sequence**; **sequence** and therapeutic applications for **human growth**-assocd. **protease inhibitor** heavy chain precursor)

IT 249916-73-2
RL: BSU (Biological study, unclassified); PRP (Properties); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(nucleotide **sequence**; **sequence** and therapeutic applications for **human growth**-assocd. **protease inhibitor** heavy chain precursor)

IT 249760-99-4, PN: WO9957140 SEQID: 3 unclaimed protein 249761-00-0, PN: WO9957140 SEQID: 4 unclaimed protein 249761-01-1, PN: WO9957140 SEQID: 5 unclaimed protein
RL: PRP (Properties)
(unclaimed protein **sequence**; **sequence** and therapeutic applications for **human growth**-assocd. **protease inhibitor** heavy chain precursor)

L5 ANSWER 6 OF 15 MEDLINE DUPLICATE 3
ACCESSION NUMBER: 2000012988 MEDLINE
DOCUMENT NUMBER: 20012988 PubMed ID: 10544273
TITLE: Multiple sites of proteolytic cleavage to release soluble forms of hepatocyte growth factor activator inhibitor type 1 from a transmembrane form.
AUTHOR: Shimomura T; Denda K; Kawaguchi T; Matsumoto K; Miyazawa K; Kitamura N
CORPORATE SOURCE: Research Center, Mitsubishi Chemical Corp., Kamoshida, Aoba-ku, Yokohama, 227-8502, Japan.
SOURCE: JOURNAL OF BIOCHEMISTRY, (1999 Nov) 126 (5) 821-8.
Journal code: 0376600. ISSN: 0021-924X.
PUB. COUNTRY: Japan
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200002
ENTRY DATE: Entered STN: 20000229
Last Updated on STN: 20000229
Entered Medline: 20000214

AB Hepatocyte growth factor activator inhibitor type 1 (HAI-1) is a Kunitz-type serine **protease inhibitor**, which was identified as a potent inhibitor of hepatocyte **growth** factor (HGF) activator from the conditioned medium of a **human** carcinoma cell line. HGF activator is a blood coagulation factor XII-like serine protease that is responsible for proteolytic activation of the inactive single chain precursor of HGF in injured tissues. The predicted **sequence** of the primary translation product of HAI-1, which has a hydrophobic **sequence** in its COOH-terminal region, suggested that HAI-1 is first produced in a membrane-associated form. In this study, we identified a transmembrane form of HAI-1 integrated in the plasma membrane of cultured cells using a monoclonal antibody against HAI-1. We also identified several soluble forms of HAI-1 in the conditioned medium of the cells, indicating that multiple sites are present in the transmembrane form of HAI-1 at which proteolytic cleavage releases the extracellular domain. At least two proteases, one of which is a metalloprotease, appear to be responsible for the release. Further, the soluble forms of HAI-1 have different inhibitory activity against HGF activator. These findings suggest that proteolytic processing plays important roles in regulation of the inhibitory activity of HAI-1.

AB Hepatocyte growth factor activator inhibitor type 1 (HAI-1) is a Kunitz-type serine **protease inhibitor**, which was identified as a potent inhibitor of hepatocyte **growth** factor (HGF) activator from the conditioned medium of a **human** carcinoma cell line. HGF activator is a blood coagulation factor XII-like serine protease that is responsible for proteolytic activation of the inactive single chain precursor of HGF in injured tissues. The predicted **sequence** of the primary translation product of HAI-1, which has a hydrophobic **sequence** in its COOH-terminal region, suggested that HAI-1 is first produced in a membrane-associated form. In this study, we identified a. . .

L5 ANSWER 7 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:682507 CAPLUS
DOCUMENT NUMBER: 129:299030
TITLE: Transgenic expression in genital tract and sexual accessory glands
INVENTOR(S): Pothier, Francois
PATENT ASSIGNEE(S): Universite Laval, Can.
SOURCE: PCT Int. Appl., 25 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9845419	A1	19981015	WO 1998-CA309	19980402
W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, GW, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG				
AU 9869140	A1	19981030	AU 1998-69140	19980402
EP 977837	A1	20000209	EP 1998-914736	19980402
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1997-825955 A2 19970403
WO 1998-CA309 W 19980402

AB The present invention relates to a method for the prodn. and secretion into animal's semen of an exogenous recombinant protein comprising the steps of: (a) producing a transgenic animal characterized by an expression system comprising a promoter specific for the genital tract or accessory

glands operatively linked to an exogenous DNA **sequence** coding for the recombinant protein through a DNA **sequence** coding for a signal peptide effective in secreting and maturing the recombinant protein in genital tract tissue; (b) collecting semen produced by said transgenic animal; and (c) isolating the exogenous recombinant protein from the semen. **Human growth** hormone cDNA was placed under control of mouse secretory **protease inhibitor** p12 promoter **sequence** in a construct that included a polyA tail and a SV40 intron for stabilization of the mRNA. Transgenic mice were generated by pronuclear injection of the construct into zygotes. After mating with transgenic males, the female vaginal plug contained 30.44 ng/mL **human** growth hormone.

AB The present invention relates to a method for the prodn. and secretion into animal's semen of an exogenous recombinant protein comprising the steps of: (a) producing a transgenic animal characterized by an expression system comprising a promoter specific for the genital tract or accessory glands operatively linked to an exogenous DNA **sequence** coding for the recombinant protein through a DNA **sequence** coding for a signal peptide effective in secreting and maturing the recombinant protein in genital tract tissue; (b) collecting semen produced by said transgenic animal; and (c) isolating the exogenous recombinant protein from the semen. **Human growth** hormone cDNA was placed under control of mouse secretory **protease inhibitor** p12 promoter **sequence** in a construct that included a polyA tail and a SV40 intron for stabilization of the mRNA. Transgenic mice were generated by pronuclear injection of the construct into zygotes. After mating with transgenic males, the female vaginal plug contained 30.44 ng/mL **human** growth hormone.

L5 ANSWER 8 OF 15 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:187651 CAPLUS
DOCUMENT NUMBER: 126:302999
TITLE: Hepatocyte growth factor activator inhibitor, a novel Kunitz-type serine protease inhibitor
AUTHOR(S): Shimomura, Takeshi; Denda, Kimitoshi; Kitamura, Akiko; Kawaguchi, Toshiya; Kito, Masahiro; Kondo, Jun; Kagaya, Shinji; Qin, Li; Takata, hiroyuki; Miyazawa, Keiji; Kitamura, Naomi
CORPORATE SOURCE: Research Center, Mitsubishi Chemical Corp., Yokohama, 227, Japan
SOURCE: J. Biol. Chem. (1997), 272(10), 6370-6376
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Hepatocyte growth factor (HGF) activator is a serine protease that is produced and secreted by the liver and circulates in the blood as an inactive zymogen. In response to tissue injury, the HGF activator zymogen is converted to the active form by limited proteolysis. The activated HGF activator converts an inactive single chain precursor of HGF to a biol. active heterodimer in injured tissue. The activated HGF may be involved in the regeneration of the injured tissue. In this study, the authors purified an inhibitor of HGF activator from the conditioned medium of a human MKN45 stomach carcinoma cell line and molecularly cloned its cDNA. The sequence of the cDNA revealed that the inhibitor has two well defined Kunitz domains, suggesting that the inhibitor is a member of the Kunitz family of serine protease inhibitors. The sequence also showed that the primary translation product of the inhibitor has a hydrophobic sequence at the C-terminal region. Inhibitory activity toward HGF activator was detected in the membrane fraction as well as in the conditioned medium of MKN45 cells. These results suggest that the inhibitor may be produced as a membrane-assocd. form and secreted by the producing cells as a proteolytically truncated form.

IT Protein **sequences**
cDNA **sequences**
(cDNA **sequence** for **human** hepatocyte **growth** factor activator inhibitor, a novel Kunitz-type serine **protease inhibitor**, and characterization of its inhibitory activity and tissue distribution)

IT 188701-87-3
RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(amino acid **sequence**; cDNA **sequence** for **human** hepatocyte **growth** factor activator inhibitor, a novel Kunitz-type serine **protease inhibitor**, and characterization of its inhibitory activity and tissue distribution)

IT 189200-91-7, Hepatocyte growth factor activator inhibitor
RL: BAC (Biological activity or effector, except adverse); BOC (Biological occurrence); PRP (Properties); BIOL (Biological study); OCCU (Occurrence)
(cDNA **sequence** for **human** hepatocyte **growth** factor activator inhibitor, a novel Kunitz-type serine **protease inhibitor**, and characterization of its inhibitory activity and tissue distribution)

IT 149885-78-9, Hepatocyte growth factor activator
RL: BPR (Biological process); BIOL (Biological study); PROC (Process)
(cDNA **sequence** for **human** hepatocyte **growth** factor activator inhibitor, a novel Kunitz-type serine **protease inhibitor**, and characterization of its inhibitory activity and tissue distribution)

IT 189176-23-6
RL: PRP (Properties)
(nucleotide **sequence**; cDNA **sequence** for **human** hepatocyte **growth** factor activator inhibitor, a novel Kunitz-type serine **protease inhibitor**, and characterization of its inhibitory activity and tissue distribution)

DUPLICATE 4

L5 ANSWER 9 OF 15 MEDLINE
ACCESSION NUMBER: 1998049419 MEDLINE
DOCUMENT NUMBER: 98049419 PubMed ID: 9389551
TITLE: The growth hormone dependent serine protease inhibitor, Spi 2.1 inhibits the des (1-3) insulin-like growth factor-I generating protease.
AUTHOR: Maake C; Yamamoto H; Murphy L J
CORPORATE SOURCE: Department of Internal Medicine, University of Manitoba, Winnipeg, Canada.
SOURCE: ENDOCRINOLOGY, (1997 Dec) 138 (12) 5630-6.
Journal code: 0375040. ISSN: 0013-7227.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199712

ENTRY DATE: Entered STN: 19980116
Last Updated on STN: 20000303
Entered Medline: 19971229

AB The conversion of insulin-like growth factor-I (IGF-I) to the biologically more active des (1-3) IGF-I variant is catalyzed by a ubiquitous protease. This proteolytic activity is inhibited by **human** alpha1-antitrypsin and soy-bean trypsin inhibitor and is up-regulated in serum and tissue extracts of hypophysectomized rats. These observations lead us to investigate whether the **growth** hormone regulated, serine **protease inhibitor**, Spi 2.1 was able to inhibit the des (1-3) IGF-I generating protease. Dihydrofolate reductase deficient Chinese hamster ovary (CHO(dhfr-ve)) cells were transfected with a rat Spi 2.1 expression vector containing the dhfr and neomycin resistance gene. Stable transfectants were selected using G418 and amplified using methotrexate. Conditioned medium from Spi 2.1 transfected CHO cells potently inhibited proteolytic activity directed against a synthetic hexa-peptide with a **sequence** identical to the N-terminal of IGF-I. In contrast conditioned medium from wild-type CHO cells had little effect. Based upon these observations we suggest that our previous finding of enhanced des (1-3) IGF-I generating protease activity in growth hormone deficient rats may be, at least partly explained by reduced levels of Spi 2.1. Furthermore, we propose that the regulation of the generation of des (1-3) IGF-I may be an additional potential site of growth hormone regulation of IGF-I action.

AB . . . the biologically more active des (1-3) IGF-I variant is catalyzed by a ubiquitous protease. This proteolytic activity is inhibited by **human** alpha1-antitrypsin and soy-bean trypsin inhibitor and is up-regulated in serum and tissue extracts of hypophysectomized rats. These observations lead us to investigate whether the **growth** hormone regulated, serine **protease inhibitor**, Spi 2.1 was able to inhibit the des (1-3) IGF-I generating protease. Dihydrofolate reductase deficient Chinese hamster ovary (CHO(dhfr-ve)) cells. . . . methotrexate. Conditioned medium from Spi 2.1 transfected CHO cells potently inhibited proteolytic activity directed against a synthetic hexa-peptide with a **sequence** identical to the N-terminal of IGF-I. In contrast conditioned medium from wild-type CHO cells had little effect. Based upon these. . .

L5 ANSWER 10 OF 15 CAPLUS COPYRIGHT 2002 ACS DUPLICATE 5
ACCESSION NUMBER: 1997:793657 CAPLUS
DOCUMENT NUMBER: 128:112241
TITLE: The anionic protease inhibitor BWI-1 from buckwheat seeds. Kinetic properties and possible biological role
AUTHOR(S): Dunaevsky, Yakov E.; Gladysheva, Inna P.; Pavlukova, Ekaterina B.; Beliakova, Galina A.; Gladyshev, Dmitry P.; Papisova, Alla I.; Larionova, Natalja I.; Belozersky, Mikhail A.
CORPORATE SOURCE: A. N. Belozersky Inst. of Physico-Chemical Biology, Moscow State Univ., Moscow, 119899, Russia
SOURCE: Physiologia Plantarum (1997), 101(3), 483-488
CODEN: PHPLAI; ISSN: 0031-9317
PUBLISHER: Munksgaard International Publishers Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Kinetic characteristics and effects on the **growth** of filamentous fungi of one of the main anionic **protease inhibitors**, BWI-1, isolated from buckwheat seeds, have been studied. The inhibition consts. of bovine trypsin, chymotrypsin and cathepsin G from **human** granulocytes with BWI-1 were found to be 1.1, 67 and 200 nM, resp. Anal. of the amino acid **sequence** of BWI-1 in the vicinity of the reactive site revealed its homol. to the potato proteinase inhibitor I family. It is suggested that the inability of BWI-1 to bind elastase of **human** granulocytes is due to the basic nature of the amino acid residue (Arg) at the P1 position in its reactive site. It was demonstrated that BWI-1 was able to suppress the germination of the spores and the growth of the mycelium of two filamentous fungi.

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L5 ANSWER 11 OF 15 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 93232057 MEDLINE
DOCUMENT NUMBER: 93232057 PubMed ID: 8473338
TITLE: Senescent WI-38 cells fail to express EPC-1, a gene induced in young cells upon entry into the G0 state.
AUTHOR: Pignolo R J; Cristofalo V J; Rotenberg M O
CORPORATE SOURCE: Center for Gerontological Research, Medical College of Pennsylvania, Philadelphia 19129.
CONTRACT NUMBER: AG00378 (NIA)
SOURCE: JOURNAL OF BIOLOGICAL CHEMISTRY, (1993 Apr 25) 268 (12) 8949-57.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M90439
ENTRY MONTH: 199305
ENTRY DATE: Entered STN: 19930604
Last Updated on STN: 19930604
Entered Medline: 19930514

AB Recently we reported the isolation of cDNAs for a number of genes that are differentially expressed between nonproliferating early (young) and late (senescent) population doubling level (PDL) WI-38 **human**, fetal lung-derived, fibroblast-like cells. We now demonstrate that one of these isolates, EPC-1 (early PDL cDNA-1), derives from an approximately 1.4-kilobase mRNA species that is expressed at a > or = 100-fold higher level in serum-starved, confluent, young versus similarly treated senescent WI-38 cells. Complete nucleotide **sequence** analysis of this cDNA confirms its identity with that of a cDNA encoding a secreted, retinal pigmented epithelium differentiation factor as well as similarity of the encoded protein with a number of mammalian serine **protease inhibitors**. We show that EPC-1 expression is associated with G0 **growth** arrest in WI-38 cells. The mRNA readily accumulates in

density-arrested and/or serum-starved young cells but not in log phase, low density young cells. In contrast, EPC-1 transcript abundance and expression of the encoded, secreted protein are both negligible in senescent WI-38 cells under all culture conditions tested. These findings support the hypothesis that senescent WI-38 cells exhibit a state of growth arrest fundamentally distinct from that of quiescent (G0) young cells.

AB . . . a number of genes that are differentially expressed between nonproliferating early (young) and late (senescent) population doubling level (PDL) WI-38 **human**, fetal lung-derived, fibroblast-like cells. We now demonstrate that one of these isolates, EPC-1 (early PDL cDNA-1), derives from an approximately. . . at a > or = 100-fold higher level in serum-starved, confluent, young versus similarly treated senescent WI-38 cells. Complete nucleotide **sequence** analysis of this cDNA confirms its identity with that of a cDNA encoding a secreted, retinal pigmented epithelium differentiation factor as well as similarity of the encoded protein with a number of mammalian serine **protease** **inhibitors**. We show that EPC-1 expression is associated with G0 **growth** arrest in WI-38 cells. The mRNA readily accumulates in density-arrested and/or serum-starved young cells but not in log phase, low. . .

DUPLICATE 7

L5 ANSWER 12 OF 15 MEDLINE
ACCESSION NUMBER: 92407628 MEDLINE
DOCUMENT NUMBER: 92407628 PubMed ID: 1326608
TITLE: Structure and chromosomal localization of the mammalian agrin gene.
AUTHOR: Rupp F; Ozcelik T; Linial M; Peterson K; Francke U; Scheller R
CORPORATE SOURCE: Howard Hughes Medical Institute, Department of Molecular and Cellular Physiology, Stanford University School of Medicine, California 94305.
SOURCE: JOURNAL OF NEUROSCIENCE, (1992 Sep) 12 (9) 3535-44. Journal code: 8102140. ISSN: 0270-6474.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-M92654; GENBANK-M92655; GENBANK-M92656; GENBANK-M92657; GENBANK-M92658; GENBANK-M92659
ENTRY MONTH: 199210
ENTRY DATE: Entered STN: 19921106
Last Updated on STN: 19921106
Entered Medline: 19921019

AB Agrin, a component of the synaptic basal lamina, has been shown to induce clustering of ACh receptors on the surface of muscle fibers. Analysis of cDNAs isolated from a rat embryonic spinal cord library demonstrated that agrin contains domains similar to regions of **protease** **inhibitors**, laminin and epidermal **growth** factor. The domain structure of agrin is further revealed here in an analysis of the agrin gene. Two additional internal repeated **sequences** are defined: one rich in cysteine residues with no homology to other proteins, and another similar to the laminin G domain, which is involved in heparin binding. Alternative RNA splicing at two positions in the gene predicts up to eight possible forms of the agrin protein. The gene (symbol AGRN/Agrn) has been assigned to chromosome 1 region pter-p32 in **human** and to mouse chromosome 4.

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DUPLICATE 8

L5 ANSWER 13 OF 15 MEDLINE
ACCESSION NUMBER: 90015171 MEDLINE
DOCUMENT NUMBER: 90015171 PubMed ID: 2507928
TITLE: Protease nexin-II, a potent antichymotrypsin, shows identity to amyloid beta-protein precursor.
AUTHOR: Van Nostrand W E; Wagner S L; Suzuki M; Choi B H; Farrow J S; Geddes J W; Cotman C W; Cunningham D D
CORPORATE SOURCE: Department of Microbiology and Molecular Genetics, University of California, Irvine 92717.
SOURCE: NATURE, (1989 Oct 12) 341 (6242) 546-9. Journal code: 0410462. ISSN: 0028-0836.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198911
ENTRY DATE: Entered STN: 19900328
Last Updated on STN: 19980206
Entered Medline: 19891117

AB Protease nexin-II (PN-II) is a **protease** **inhibitor** that forms SDS-resistant inhibitory complexes with the epidermal **growth** factor (EGF)-binding protein, the gamma-subunit of nerve growth factor, and trypsin. The properties of PN-II indicate that it has a role in the regulation of certain proteases in the extracellular environment. Here we describe more of the amino-acid **sequence** of PN-II and its identity to the deduced **sequence** of the amyloid beta-protein precursor (APP). Amyloid beta-protein is present in neuritic plaques and cerebrovascular deposits in individuals with Alzheimer's disease and Down's syndrome. A monoclonal antibody against PN-II (designated mAbP2-1) recognized PN-II in immunoblots of serum-free culture medium from **human** glioblastoma cells and neuroblastoma cells, as well as in homogenates of normal and Alzheimer's disease brains. In addition, mAbP2-1 stained neuritic plaques in Alzheimer's disease brain. PN-II was a potent inhibitor of chymotrypsin with an inhibition constant Ki of 6 x 10(-10)M. Together, these data demonstrate that PN-II and APP are probably the same protein. The regulation of extracellular proteolysis by PN-II and the deposition of at least parts of the molecule in senile plaques is consistent with previous reports that implicate altered proteolysis in the pathogenesis of Alzheimer's disease.

AB Protease nexin-II (PN-II) is a **protease** **inhibitor** that forms SDS-resistant inhibitory complexes with the epidermal **growth** factor (EGF)-binding protein, the gamma-subunit of nerve growth factor, and trypsin. The properties of PN-II indicate that it has a role in the regulation of certain proteases in the extracellular environment. Here we describe more of the amino-acid **sequence** of PN-II and its

identity to the deduced **sequence** of the amyloid beta-protein precursor (APP). Amyloid beta-protein is present in neuritic plaques and cerebrovascular deposits in individuals with Alzheimer's. . . . disease and Down's syndrome. A monoclonal antibody against PN-II (designated mAbP2-1) recognized PN-II in immunoblots of serum-free culture medium from **human** glioblastoma cells and neuroblastoma cells, as well as in homogenates of normal and Alzheimer's disease brains. In addition, mAbP2-1 stained. . .

L5 ANSWER 14 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1987:315792 BIOSIS
DOCUMENT NUMBER: BA84:35299
TITLE: STUDY OF A GROWTH HORMONE-REGULATED PROTEIN SECRETED BY RAT HEPATOCYTES COMPLEMENTARY DNA CLONING ANTI-PROTEASE ACTIVITY AND REGULATION OF ITS SYNTHESIS BY VARIOUS HORMONES.
AUTHOR(S): LE CAM A; PAGES G; AUBERGER P; LE CAM G; LEOPOLD P; BENAROUS R; GLAICHENHAUS N
CORPORATE SOURCE: UNITE INSERM 145, FAC. MED. 06034 NICE, FR.
SOURCE: EMBO (EUR MOL BIOL ORGAN) J, (1987) 6 (5), 1225-1232.
CODEN: EMJODG. ISSN: 0261-4189.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB GHR-P63 ('growth hormone-regulated protein 63,000 daltons') is an acidic glycoprotein secreted by rat hepatocytes whose synthesis is abolished upon hypophysectomy. The sequence of its mRNA including the entire coding and 3' untranslated regions was determined from a nearly full-length .lambda.gt11-cDNA clone. The sequence contained two ATGs in frame giving rise to two overlapping coding regions which could encode precursor polypeptides of 416 and 406 amino acid residues (Mrs = 46549 and 45371). These potential translation initiation codons apppeared to be functional both in vitro and in intact cells since two precursors of the correct size were immunoprecipitated as products of mRNA translation. The unglycosylated precursors were converted into intermediate intracellular forms of about 56,000 daltons containing N-linked oligosaccharide side chains and thereafter into the secretory form of .simeq. 63,000 daltons. Strong sequence homologies, both at the nucleotide and the amino acid levels were found between GHR-P63 and several serum protease inhibitors, more particularly mouse contrapsin and human .alpha.1-antichymotrypsin. In agreement with sequence data, GHR-P63 purified from rat blood by affinity chromatography was found to carry an anti-trypsin activity. GHR-P63 mRNA, virtually undetectable in hepatocytes from hypophysectomized rats, could be hormonally re-induced to subnormal levels both in vivo by treating animals with hormones and in vitro by incubating the defective cells with hormones. Growth hormone was absolutely requied but had a weak effect when used alone. Glucocorticoids which had no effect when used per se, strongly potentiated the action of growth hormone. Nuclear run-off experiments suggest that hormones regulated GHR-P63 mRNA levels by acting mostly, if not exclusively, on gene transcription.
IT Miscellaneous Descriptors
HUMAN ALPHA-1 ANTICHYMOTRYPSIN MOUSE CONTRAPSIN MESSENGER RNA COMPLEMENTARY DNA NUCLEOTIDE **SEQUENCE** SERUM **PROTEASE INHIBITORS** HYPOPHYSECTOMY **GROWTH** HORMONE

DUPLICATE 9

L5 ANSWER 15 OF 15 MEDLINE
ACCESSION NUMBER: 87156686 MEDLINE
DOCUMENT NUMBER: 87156686 PubMed ID: 2435284
TITLE: Amino-terminal sequence of a large form of basic fibroblast growth factor isolated from human benign prostatic hyperplastic tissue.
AUTHOR: Story M T; Esch F; Shimasaki S; Sasse J; Jacobs S C; Lawson R K
CONTRACT NUMBER: AM31063 (NIADDK)
SOURCE: BIOCHEMICAL AND BIOPHYSICAL RESEARCH COMMUNICATIONS, (1987 Feb 13) 142 (3) 702-9.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198704
ENTRY DATE: Entered STN: 19900303
Last Updated on STN: 19970203
Entered Medline: 19870401
AB Homogenization of **human** benign prostatic hyperplastic tissue in high ionic strength alkaline buffer containing **protease inhibitors** resulted in the isolation of a 17,400 molecular weight **growth** factor. When tissue was homogenized in ammonium sulfate at pH 4.5 without **protease inhibitors** a smaller, 16,600 dalton, **growth** factor was isolated. Both **growth** factors reacted with antisera against synthetic peptides whose **sequences** corresponded to the amino-terminal (1-12), Internal (33-43) and carboxyl-terminal (135-145) portions of basic fibroblast growth factor (bFGF). This suggested that the smaller growth factor was not a truncated form of (1-146) bFGF and that the larger growth factor may contain additional **sequences**. Amino-terminal sequencing showed the larger growth factor to have the **sequence**: Ala-Ala-Gly-Ser-Ile-Thr-Thr-Leu-Pro-Ala-Leu-Pro-Glu-Asp-Gly-Gly-Ser-Gly-Ala-Phe-Pro-. These results show that the larger growth factor is an 8 amino acid extended from of (1-146) bFGF and it is likely that the smaller growth factor is a proteolytic cleavage product of the larger growth factor produced during the extraction procedure.
AB Homogenization of **human** benign prostatic hyperplastic tissue in high ionic strength alkaline buffer containing **protease inhibitors** resulted in the isolation of a 17,400 molecular weight **growth** factor. When tissue was homogenized in ammonium sulfate at pH 4.5 without **protease inhibitors** a smaller, 16,600 dalton, **growth** factor was isolated. Both **growth** factors reacted with antisera against synthetic peptides whose **sequences** corresponded to the amino-terminal (1-12), Internal (33-43) and carboxyl-terminal (135-145) portions of basic fibroblast growth factor (bFGF). This suggested that. . . smaller growth factor was not a truncated form of (1-146) bFGF and that the larger growth factor may contain additional **sequences**. Amino-terminal sequencing showed the larger growth factor to have the **sequence**: Ala-Ala-Gly-Ser-Ile-Thr-Thr-Leu-Pro-Ala-Leu-Pro-Glu-Asp-Gly-Gly-Ser-Gly-Ala-Phe-Pro-. These results show that the larger growth factor is an 8 amino acid extended from of (1-146) bFGF. . .

=> dis his

(FILE 'HOME' ENTERED AT 13:52:16 ON 12 JUN 2002)

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     FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 13:52:33 ON 12 JUN 2002
L1        70323 S (PROTEASE (1N) INHIBITOR?)
L2          912 S L1 (10N) GROWTH
L3          212 S L2 (P) HUMAN?
L4           35 S L3 (P) SEQUENCE?
L5           15 DUP REM L4 (20 DUPLICATES REMOVED)

=> end
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:Y                                  SINCE FILE     TOTAL
COST IN U.S. DOLLARS                                     ENTRY   SESSION
                                                         41.48     41.69

FULL ESTIMATED COST

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)            SINCE FILE     TOTAL
                                                         ENTRY   SESSION
                                                         -3.10     -3.10

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STN INTERNATIONAL LOGOFF AT 14:06:04 ON 12 JUN 2002
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18
      (FILE 'HOME' ENTERED AT 13:24:01 ON 12 JUN 2002)

      FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 13:24:18 ON 12 JUN 2002
L1          3870 S HILLMAN J?/AU OR GUEGLER K?/AU OR PATTERSON C?/AU
L2            80 S L1 AND PROTEASE?
L3            15 S L2 AND GROWTH?
L4             7 DUP REM L3 (8 DUPLICATES REMOVED)
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(FILE 'HOME' ENTERED AT 13:52:16 ON 12 JUN 2002)

FILE 'MEDLINE, CAPLUS, EMBASE, BIOSIS' ENTERED AT 13:52:33 ON 12 JUN 2002

L1 70323 S (PROTEASE (1N) INHIBITOR?)
L2 912 S L1 (10N) GROWTH
L3 212 S L2 (P) HUMAN?
L4 35 S L3 (P) SEQUENCE?
L5 15 DUP REM L4 (20 DUPLICATES REMOVED)